



Caries Prevention and Reversal Based on the Caries Balance

John D.B. Featherstone, MSc, PhD¹

Abstract

The science behind caries prevention and reversal is well understood. A recent clinical trial has confirmed that reducing caries risk results in a reduction in dental decay. Dental caries progression or reversal depends upon the balance between demineralization and remineralization and can be visualized for clinical purposes as the “caries balance.” This balance is determined by the relative weights of the sums of pathological factors and protective factors. A structured caries risk assessment should be carried out based upon the concept of the caries balance. Following the risk assessment, a treatment plan is devised which leads to the control of dental caries for the patient. The balance between pathological and preventive factors can be swung in the direction of caries intervention and prevention by the active role of the dentist and his/her auxiliary staff. (*Pediatr Dent* 2006;28:128-132)

KEYWORDS: DENTAL CARIES, DEMINERALIZATION, REMINERALIZATION, PREVENTIVE DENTISTRY

Introduction

Many of the details of the dental caries process have been known for a long time. Even though new research findings are continually being published on various aspects of dental caries such as the microbiology of caries, the biofilm, demineralization and remineralization, fluoride applications, dietary components, saliva, and fluoride-containing dental materials, we seem to still be unable to embrace the science and fully implement it to reduce the level of dental caries in the population.¹ The most recent NHANES survey² for the period 1999-2002 shows that caries levels at all ages are still a major problem, especially among teenagers. With all of the scientific evidence that we have about caries prevention, including many clinical trials of various agents, why is it that dental practice has not embraced the concepts of prevention and intervention? For example, a review by Anderson et al.³ laid out step-by-step procedures that would reduce dental decay in individuals. This excellent article was never embraced by the profession. Anusavice^{4, 5} provided an update and reached out especially to the dental materials community. Numerous publications during the 1980s summarized knowledge about fluoride action, saliva, downward trends in caries in various countries around the world where fluoride toothpaste was used, and other aspects of caries prevention. The knowledge is in the literature. It is time for us to translate the laboratory and clinical findings into actual practice and to deal with the disease that is dental caries. It is time for a paradigm shift.

¹Dr. Featherstone is professor, Department of Preventive and Restorative Dental Sciences, University of California, San Francisco, Calif. Correspond with Dr. Featherstone at jdbf@ucsf.edu

The caries balance concept

The concept of the caries balance was first published by Featherstone in 1999⁶ in an attempt to simplify the key factors involved in dental caries progression or reversal and make them readily applicable in clinical practice and easily understandable for the patient. The concept was included in a review¹ that provided a simple summary of the science of caries prevention. Most recently, the concept was embraced as part of a two-day consensus meeting in California to review the relevant literature on caries prevention and propose practical means for caries management by risk assessment in the dental office and in community health settings.^{7,8} Each of the review papers, the consensus statement, and the caries risk assessment forms are available on the web at www.cdafoundation.org/journal.

The caries process can easily be visualized as a balance between pathological factors and protective factors as shown in the figure. If the pathological factors outweigh the protective factors, then caries progresses. The balance is presented here with three key factors on each side. More detail could be added, but in our experience simpler is better to enable students and practitioners to grasp and use the balance as a basis for understanding caries progression or reversal in individual patients. Step-by-step instructions for use in the clinical environment have been published elsewhere.^{9,10} In summary, the pathological and protective factors are described as follows:

Pathological factors

1. Cariogenic bacteria. Any bacteria in the dental plaque that produce acids (so-called acidogenic bacteria) must be considered cariogenic. Studies have shown that com-

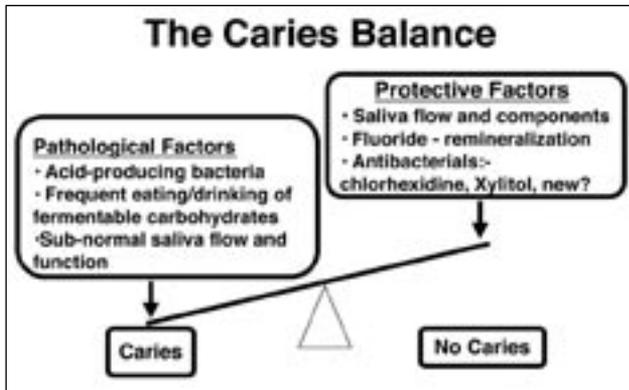


Figure. Conceptual illustration of the caries balance concept. If the pathological factors outweigh the protective factors, caries progresses.

binations of acetic and lactic acid are more damaging than lactic acid alone.¹¹ This means that combinations of species such as *Streptococcus mutans*, *Streptococcus sobrinus* (the mutans streptococci group), and the lactobacilli species all contribute to caries progression, perhaps even more than just additively.¹ Other acid-producing species have been identified and will also add to the acid challenge. Several bacterial species are also aciduric, that is they can live in acid, which adds to their virulence.¹² Attempts to deal with the bacterial challenge must target more than one species. It is very important that we add antibacterial treatment as part of the regimen for high caries risk individuals.

2. Fermentable carbohydrates. The frequent ingestion of fermentable carbohydrates is well known as a necessary factor for caries initiation and progression. The carbohydrates involved include sucrose, glucose, fructose, cooked starch, and potentially any carbohydrate that any of the acidogenic species can metabolize.
3. Salivary dysfunction. Saliva and its components are essential to the maintenance of oral health. Markedly reduced saliva flow, and consequently reduced delivery of all the beneficial components of saliva, immediately places a person at high risk for caries progression.¹³

Protective factors

1. Saliva components and flow. Saliva flow helps to flush carbohydrates from the plaque, while at the same time providing buffers against the plaque-derived acids. Saliva also provides proteins and lipids to form the protective pellicle, proteins to maintain calcium and phosphate in a supersaturated state, and antibacterial proteins. Saliva also functions as a carrier of fluoride to the plaque fluid.^{13,14}
2. Fluoride from extrinsic sources, and calcium and phosphate from saliva. Fluoride from extrinsic sources, such as fluoride-containing dental products, inhibits demineralization if present at the crystal surface at the time of acid challenge. Fluoride, calcium, and

phosphate together provide the key ingredients for remineralization, which is the natural repair process for the early carious lesion. Remineralized enamel or dentin is more resistant to subsequent acid challenges as described in detail elsewhere.¹⁵

3. Antibacterial therapy. While saliva provides some natural antibacterial therapy, this is insufficient if the pathological factors outweigh the protective factors. In cases where the bacterial challenge is high and the person is at high risk for future caries, then additional antibacterial treatment is necessary to allow fluoride and remineralization to balance the challenge. Chlorhexidine can effectively reduce mutans streptococci levels in the plaque biofilm, but it is much less effective against the lactobacilli species.¹⁶ Xylitol, a noncariogenic sweetener, also interferes with bacterial adherence and transmission.¹⁷ Further information is available at www.cdafoundation.org/journal.

The above three pathological factors and three protective factors are not exclusive, but are the most important factors in assessing future caries risk and identifying why a person has caries or can be expected to show caries continue progression. Additional items including tooth morphology, oral hygiene, sealant application, and fluoride-releasing dental materials can be added, but the 3-vs-3 concept should be the first step in providing a risk assessment and an understanding of the caries process as it is progressing in an individual patient. It is easy to remember and easy to apply chairside as part of the “detective-like” investigation that should be done for each patient to assess the likelihood of future caries, and the reasons for existing lesions.

Caries management by risk assessment

Caries risk assessment has been discussed for decades and several attempts have been made to produce risk assessment tools. A recently completed clinical trial has illustrated that caries can be managed by risk assessment and chemical therapy.¹⁸ A brief summary is given here. Although the study was done in high caries risk adults, the results are expected to apply equally well to children and especially to adolescents. The overall objective of the study was to provide clinical evidence that the concept of caries management by risk assessment really works, and that tipping the balance from pathological factors predominating to protective factors predominating results in less clinical caries lesions.

Adult subjects, 18 years and older, who had 1 to 7 cavitated carious lesions at baseline were randomized to either a “conventional care” control group or to a preventive intervention group. Because they had frank cavities at enrollment, these subjects were considered to be at high risk for future caries, which was shown to be correct in the control group. Saliva samples were taken at least every 6 months for MS, lactobacilli and fluoride evaluation. Caries exams were conducted at baseline and 2 years after all restorations of the original frank cavities were completed. Chlorhexidine gluconate 0.12% rinse was used in the intervention group

based upon bacterial challenge, together with fluoride dentifrice daily, and 0.05% NaF rinse. Caries risk status (low, moderate, high) was assessed every 6 months, based on combined bacterial counts and fluoride assays. Mutans streptococci counts fell markedly in the intervention group as a result of the chlorhexidine therapy. Reduction in caries risk status was strongly correlated with reduction in numbers of decayed teeth. We concluded that targeted combined antibacterial and fluoride therapy beneficially altered caries risk status and caries incidence.

Clinical implications

The clinical implications of the above information are considerable. The caries balance concept can be used to drive diagnosis and clinical treatment to arrest or reverse the disease of dental caries in individuals and also to drive treatments in community clinics. What does this statement mean in terms of what to do?

The bottom line principles are:

1. reduce the pathological factors;
2. increase the protective factors.

By doing the above we tip the balance (Figure) and bring the patient under control. A simple caries risk assessment should be done at the baseline examination, and at all subsequent periodic oral exams. Following a traditional clinical examination, it only takes a few minutes to go through the key pathological and protective factors and decide whether the patient is at high, moderate, or low risk. One suggested form to use as a guide has been published as a result of the California meeting mentioned above⁸ (www.cdafoundation.org/journal). The form is fully compatible with the concepts presented in the American Academy of Pediatric Dentistry CAT form.¹⁹ The treatment plan is then designed to address each of the items identified. For example, the following are common caries risk indicators or risk factors that can readily be dealt with:

Common caries risk indicators and risk factors

1. Visible cavitation or caries into the dentin by radiograph. In this case it is very likely that the patient has high levels of MS and/or lactobacilli. A bacterial test is immediately indicated to provide a baseline for measuring the success of antibacterial treatments. If a patient has ≥ 1 frank cavities, antibacterial therapy is essential to bring the bacteria levels down and give fluoride a chance to work effectively. Antibacterial therapy should be started immediately and continued while restorative work is in progress, and until the bacterial levels are under control as evidenced by culture testing. At the time of writing, .12% chlorhexidine gluconate mouthrinse once a day for 1 week every month for at least 6 months is suggested for older children and adults (see below).
2. Caries lesions restored in the last three years. If the patient has had recent lesions restored, this is also a good indicator that new caries lesions will occur in the

immediate future. Bacterial testing may indicate that antibacterial therapy is needed.

3. Readily visible heavy plaque on the teeth. This indicates poor oral hygiene and/or prolific plaque growth by the individual and is an indirect indicator that there are likely to be high levels of cariogenic bacteria.
4. Frequent between meal snacks of fermentable carbohydrates. If the patient is snacking greater than 3 times daily between meals on foods or beverages that contain sucrose, glucose, fructose, or cooked starch (cookies or bread), this increases the acid challenge to the teeth to a high level. Xylitol-containing gum or mints should be recommended as a substitute for these snacks.
5. Saliva reducing factors. If a patient is taking hyposalivatory medications such as tranquilizers or mood-altering medications, saliva flow will be reduced. Patients taking multiple medications are at increased risk. If a patient is suffering from cancer to the head and neck and receiving radiation treatment, this may also severely impair salivary function. Saliva flow rate can be measured by having the patient chew and spit into a measuring cup and calculate the number of milliliters (mL) per minute. A value less than 0.7 mL/minute is low, whereas 1 to 4 mL/minute is normal.
6. Fixed or removable appliances in the mouth. The presence of fixed or removable appliances in the mouth such as orthodontic brackets or removable partial dentures leads to undue accumulation of plaque and an increase in the percent of cariogenic bacteria. These appliances will generally place the patient at high risk of new caries lesions in the future.

Daily oral hygiene

Twice daily brushing with a fluoride toothpaste/dentifrice should be recommended to all patients, regardless of risk level. Not only will this reduce the plaque level in the mouth, but most importantly it will provide therapeutic levels of fluoride to the mouth on a regular basis. Fluoride is retained in the mouth for several hours subsequent to brushing with a fluoride toothpaste, and application twice daily is an effective therapeutic measure.²⁰

Additional fluoride

For moderate and high-risk patients, additional fluoride should be recommended depending upon on the assessment of the likelihood of compliance. An over-the-counter fluoride rinse containing .05% sodium fluoride is very effective if used twice daily in addition to a fluoride toothpaste.²¹ For high-risk patients over the age of 6 years, a prescription brush-on dentifrice with a high concentration of fluoride content, such as 5,000 ppm, should be prescribed as a replacement for their regular dentifrice. Fluoride varnish is also a very effective, easily applied product that enhances remineralization. Application in the dental office reduces the need for individual patient compliance with home use products. Fluoride varnish can also be used effectively for young children under the age of 5 years, with 3 applications per year being very effective.²²

Chewing gum

The use of chewing gum can be recommended for high-risk patients, especially those with low saliva flow. The use of chewing gum stimulates the saliva and thereby enhances remineralization. Chewing gum or mints that do not contain fermentable carbohydrates (sugar-free) should be recommended. Xylitol-containing gums and mints are now readily available from several suppliers and should be recommended.

Antibacterial rinse

High-risk patients who have medium to high levels of MS, lactobacilli, or both should be placed on an antibacterial rinse. Currently the most effective antibacterial rinse against cariogenic bacteria is chlorhexidine gluconate, 0.12%. Older children or adults should rinse once daily with 10 mL for 1 minute at bedtime for 1 week.¹⁸ This should be done for 1 week every month for up to 6 months. At that time another bacterial test should be done. If used only 1 week per month, staining should be a minimal issue, based on our experience with this regimen. Compliance is also a major issue with this product, which is why we recommend use for only 1 week at a time and repeating monthly rather than 2 weeks every 3 months, which has been the recommendation in past literature.¹⁶ New antibacterials are expected to be available in the very near future and at that time their use will be recommended.

Summary and Conclusions

There is now strong scientific evidence that we can use the caries balance concept in treating caries as a disease for all ages. Reducing the pathological factors and enhancing the protective factors leads to reduction in risk and subsequent reduction in caries. We must incorporate these concepts into everyday clinical practice, with interventive therapy coming before as well as during restorative work. A paradigm shift is required to make this work. The steps are:

- 1) Detect carious lesions early enough to reverse or prevent progression;
- 2) Assess the caries risk and use this assessment to drive the treatment plan;
- 3) Daily fluoride dentifrice home use for all;
- 4) In high-risk patients start antibacterial therapy immediately. Perform a baseline bacterial culture as a benchmark against which to measure effectiveness of the therapy over time, and as a measure of compliance;
- 5) For moderate and high-risk patients employ additional fluoride therapy such as fluoride varnish or high concentration fluoride dentifrice or gel;
- 6) Add xylitol gum or mints as a substitute for between meal snacks, and most importantly to enhance remineralization and supplement antibacterial therapy;
- 7) Use minimally invasive restorative procedures to restore tooth function.

References

1. Featherstone JDB. The science and practice of caries prevention. *J Am Dent Assoc* 2000;131:887-899.
2. Beltran-Aguilar ED, Barker LK, Canto MT, Dye BA, Gooch BF. (2005) Surveillance for Dental Caries, Dental Sealants, Tooth Retention, Edentulism, and Enamel Fluorosis-United States, 1988-1994 and 1999-2002. *Surveillance Summaries, Centers for Disease Control and Prevention*, 54(No. SS-3):1-43.
3. Anderson MH, Bales DJ, Omnell K-A. Modern management of dental caries: the cutting edge is not the dental burr. *J Am Dent Assoc* 1993;124:37-44.
4. Anusavice KJ. Treatment regimens in preventive and restorative dentistry. *J Am Dent Assoc* 1995; 126:727-743.
5. Anusavice KJ. Efficacy of nonsurgical management of the initial caries lesion. *J Dental Educ* 1997; 61:895-905.
6. Featherstone JDB. Prevention and reversal of dental caries: role of low level fluoride. *Community Dent Oral Epidemiol* 1999;27:31-40.
7. Featherstone JDB. The caries balance: Contributing factors and early detection. *CDA Journal* 2003; 31:129-133.
8. Featherstone JDB, Adair SM, Anderson MH, et al. Caries Management by risk assessment: Consensus statement, April 2002. *CDA Journal* 2003; 31:257-269.
9. Featherstone JDB. Tipping the scales towards caries control. *Dimensions of Dental Hygiene* 2004; 2:20-27.
10. Featherstone JDB. The caries balance. *Dimensions of Dental Hygiene* 2004;2:14-18.
11. Featherstone JDB, Rodgers BE. The effect of acetic, lactic and other organic acids on the formation of artificial carious lesions. *Caries Res* 1981;15:377-385.
12. Loesche WJ. Role of *Streptococcus mutans* in human dental decay. *Microbiological Reviews* 1986; 50:353-380.
13. Mandel ID. The role of saliva in maintaining oral homeostasis. *J Am Dent Assoc* 1989;119:298-304.
14. Lamkin MS, Oppenheim FG. Structural features of salivary function. *Critical Reviews in Oral Biology and Medicine* 1993;4:251-259.
15. Ten Cate JM, Featherstone JDB. Mechanistic aspects of the interactions between fluoride and dental enamel. *CRC Critical Reviews in Oral Biology* 1991; 2:283-296.
16. Krasse B. Biological factors as indicators of future caries. *Int Dent J* 1988;38:219-225.
17. Lynch H, Milgrom P. Xylitol and dental caries: An overview for the clinician. *J Calif Dent Assoc* 2003;31:205-209.
18. Featherstone JDB, Gansky SA, Hoover CI, et al. A randomized clinical trial of caries management by risk assessment. *Caries Res* 2005;39:295.

19. AAPD. Caries-risk Assessment Tool (CAT). Available at: www.aapd.org/media/policies.asp. Accessed March 1, 2006.
20. Zero DT, Raubertas RF, Pedersen AM, Fu J, Hayes AL, Featherstone JDB. Fluoride concentrations in plaque, whole saliva and ductal saliva after applications of home-use fluoride agents. *J Dent Res* 1992; 71:1768-1775.
21. O'Reilly MM, Featherstone JDB. De- and remineralization around orthodontic appliances: an in vivo study. *Am J Orthod* 1987;92:33-40.
22. Weintraub JA, Ramos-Gomez F, Jue B, Shain S, Hoover CI, Featherstone JDB, Gansky SA. Fluoride varnish efficacy to prevent early childhood caries. *J Dent Res* 2006;85:172-176.

Abstract of the Scientific Literature



Emdogain Does Not Prevent Root Resorption in Reimplanted Teeth

The aim of this study was to evaluate the effectiveness of Emdogain on the periodontal healing of avulsed teeth prior to and after the onset of ankylosis. The present study was performed at the Trauma Centre of the University Hospital of Copenhagen, Denmark. Sixteen teeth in 7 patients (mean age=12.4) comprised the new trauma study group, whereas 11 teeth in 7 patients (mean age=12.7) were enrolled in the previous ankylosis group. Newly traumatized teeth sustained a combined wet and dry extraoral time of 25 to 270 minutes prior to reimplantation. Previously ankylosed teeth were diagnosed as such through their metallic tone on percussion testing. Newly avulsed teeth and those extracted upon evidence of ankylosis were treated with extraoral retrograde root canal therapy with gutta percha and IRM if not previously completed. Emdogain was applied to the root surface and into the saline rinsed alveolar socket. The teeth were splinted for 7 days and then a follow-up program was undertaken at 3 weeks and 2, 6, and 12 months post reimplantation. In the new trauma group, ankylosis occurred in 50% of teeth after 2 months and all teeth by 1 year post injury. In the previously ankylosed group, 72% were ankylosed again by 2 months and all teeth by 4 months post reimplantation.

Comments: Emdogain has been reported to be effective in the treatment of marginal periodontitis, where root surfaces are necrotic or injured. This study shows that Emdogain displays minimal effectiveness in cessation or prevention of ankylosis secondary to tooth reimplantation. Ankylosis of reimplanted teeth is a grave complication leading to infraocclusion and possible tooth loss. Dentists must inform their patients of this sequelae at the time of injury and maintain a rigorous follow-up regimen to address these complications. **JMK**

Address correspondence to Dr. J.O. Andreasen, Department of Oral and Maxillofacial Surgery, University of Copenhagen (Rigshospitalet), Blegdamsvej 9, Copenhagen-2100, Denmark.

Schjott M, Andreasen JO. Emdogain does not prevent progressive root resorption after replantation of avulsed teeth: a clinical study. *Dent Traumatol* 2005;21:46-50.

15 references